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SCIENTIFIC INVESTIGATIONS

Sleep bruxism is highly prevalent in adults with obstructive sleep apnea: a large-scale polysomnographic study

Deshui Li, DDS, MSD[1,2,3]; Boyuan Kuang, DDS, MSD[1,2,4]; Frank Lobbezoo, DDS, PhD[1]; Nico de Vries, MD, PhD[1,5,6]; Antonius Hilgevoord, MD, PhD[2]; Ghizlane Aarab, DDS, PhD[1]

[1]Department of Orofacial Pain and Dysfunction, Academic Centre for Dentistry Amsterdam, University of Amsterdam and Vrije Universiteit Amsterdam, Amsterdam, The Netherlands; [2]Department of Clinical Neurophysiology, OLVG West, Amsterdam, The Netherlands; [3]Department of Orthodontics, School and Hospital of Stomatology, Shandong University, Jinan, China; [4]Taikang Bybo Dental, Shanghai, China; [5]Department of Otorhinolaryngology, OLVG West, Amsterdam, The Netherlands; [6]Faculty of Medicine and Health Sciences, Department of Otorhinolaryngology, Head and Neck Surgery, Antwerp University Hospital, Edegem, Belgium

Study Objectives: The aim was to determine the prevalence and risk factors of sleep bruxism (SB) and to investigate the relationships between SB episodes, arousals, and respiratory events in adults with obstructive sleep apnea (OSA).

Methods: This prospective study included 914 adults with OSA (305 females, 609 males; age = 53 years [interquartile range = 17]; apnea-hypopnea index = 13.9 events/h [interquartile range = 21]). The diagnosis of SB was made when the rhythmic masticatory muscle activity (RMMA) index was at least 2 episodes/h of sleep based on a full polysomnographic recording. Binary logistic regression was performed to identify risk factors for SB. Network analysis was performed to determine the relations between RMMA, respiratory event, sleep arousal, and other factors. Further, the percentage of RMMA time-related to arousal was calculated.

Results: The prevalence of SB in adults with OSA was 49.7%. Male sex, lower body mass index, and higher percentage of N1 sleep increased the odds of having SB (odds ratios = 1.425, 0.951, and 1.032, respectively; all $P < .05$). Network analysis showed that there were no direct associations between RMMA and apnea-hypopnea index, nor between RMMA and arousal, although 85.7% of RMMA was time-related to arousals.

Conclusions: Nearly half of adults with OSA have comorbid SB. Male sex, lower body mass index, and a higher percentage of light sleep increase the risk of having SB. Although RMMAs do not directly correlate with respiratory events and arousals, most RMMAs are time-related to arousals in adults with OSA.

Clinical Trial Registration: Registry: Netherlands Trial Register; Name: A Large Sample Polysomnographic Study on Sleep Bruxism; URL: https://trialsearch.who.int/Trial2.aspx?TrialID=NL8516; Identifier: NL8516.

Keywords: sleep bruxism, obstructive sleep apnea, polysomnography, prevalence, risk factor, arousal, respiratory event

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BRIEF SUMMARY

Current Knowledge/Study Rationale: Sleep bruxism is a rhythmic masticatory muscle activity during sleep. Although previous studies suggested that sleep bruxism may play a protective role in adults with obstructive sleep apnea (OSA), the prevalence of sleep bruxism in adults with OSA and the associations between sleep bruxism, arousals, and respiratory events in OSA are inconclusive.

Study Impact: This large-scale polysomnographic study showed that sleep bruxism is highly prevalent in adults with OSA, especially in males with a low body mass index. Although sleep bruxism events did not directly correlate with respiratory events and arousals, most of them were time-related to arousals in adults with OSA. Future studies are needed to determine the effects of OSA treatments on sleep bruxism.

INTRODUCTION

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder characterized by complete (apnea) or partial (hypopnea) collapse of the upper airway, which commonly leads to sleep arousal and oxygen desaturation.[1] Patients with OSA often complain of excessive daytime sleepiness, morning headache, and snoring.[1] Of the general population, 9–38% experience OSA.[2] Male sex, older age, and overweight or obesity are risk factors for OSA.[2] Conversely, OSA can be an independent risk factor for many other medical conditions, such as diabetes, hypertension, stroke, depression, and sleep bruxism (SB).[3,4]

SB is characterized by rhythmic masticatory muscle activity (RMMA) during sleep, which manifests as clenching or grinding of the teeth and/or bracing or thrusting of the mandible.[5] In the general population, the prevalence of SB is approximately 13%.[6] The consequences of SB vary from person to person, and also from negative to positive. The negative consequences include tooth wear or fracture, orofacial pain, temporal mandibular disorders, and failure of dental prostheses and oral implants.[7,8] The suggested positive effects include clearing esophageal acid and lubricating the upper airway by promoting saliva secretion[9] and reinforcing the upper airway after respiratory events in OSA.[10]

Mandibular advancement appliances are considered a primary treatment option in mild to moderate OSA.[11] As a consequence of their SB, however, adults with OSA may break their mandibular advancement appliances during sleep and/or develop temporomandibular disorders.[12,13] Therefore, it is clinically relevant to determine the prevalence of and risk factors for SB in adults with OSA. The prevalence of SB in adult patients with OSA is probably much higher than that in the general population.[14] However, since previous studies used different methods for diagnosing SB (eg, self-report, clinical inspection, and polysomnography [PSG]) and included limited study samples, the occurrence rate of SB in the OSA population ranges widely—from 26% to 100%.[15–18] In addition, age, sex, body mass index (BMI), sleep stages, arousals, respiratory factors (eg, oxygen desaturation), and some diseases or disorders (eg, insomnia, periodic leg movements during sleep) have all been reported to be associated with SB in different studies.[14,19–22] However, although only a few previous studies took all of these factors into consideration in a single study, the results of those previous studies were inconsistent. Tan et al[16] showed that respiratory arousal increases the odds of having SB, whereas apnea-hypopnea index (AHI) and oxygen desaturation index had no effect on SB in 147 adults with OSA. However, the diagnosis of SB in this study was made when the RMMA index was more than 4 episodes/h. Thereof, low-frequency SB (RMMA ≥ 2 episodes/h) was included in the control group,[16] which makes this study difficult to interpret. In addition, Martynowicz et al[18] showed that higher AHI, male sex, and diabetes increased the RMMA index in a subgroup with AHI < 30 events/h, whereas sleep arousal did not have any effect on the RMMA index. However, this study was performed in 110 adults with possible OSA, which included cases without OSA. Therefore, a large-scale PSG study that includes all potential risk factors is needed to determine the prevalence of and risk factors for SB in adults with OSA. Based on previous findings, we hypothesized that SB is highly prevalent in adults with OSA, and that aging, obesity, and arousal will show a significant association with SB.

In addition, although OSA is considered a risk for SB, the cause-and-effect relationship between them is still inconclusive. Currently, the genesis of RMMA may involve, among others, several physiological factors that are related to OSA, such as sleep arousals and respiratory events.[7] Several studies reported that most RMMAs occur shortly after respiratory events in OSA, suggesting that SB may be secondary to respiratory events and play a protective role against OSA by restoring the upper airway.[4,23] However, other studies indicated that masticatory muscle activities after respiratory events are nonspecific orofacial activities and not RMMA.[24] Further, some studies reported that RMMAs occurring after respiratory events are more like motor responses to respiratory arousals rather than to the preceding respiratory events per se.[25,26] Nonetheless, other studies showed that arousal has only a weak association with RMMA in OSA, and that it only acts as a permissive window for the occurrence of RMMA.[16,24,27] Considering all of this evidence, the associations between RMMAs, arousals, and respiratory events in OSA are still inconclusive and need further studies to be clarified. Based on previous findings, we hypothesized that RMMA is not correlated with respiratory events but rather with sleep arousals. Additionally, as mentioned above, the associations between RMMA, respiratory events, and arousals are interactive; thus, the relationship between them may be direct or indirect. Therefore, a novel approach, such as network analysis, would be suitable to show all associations between included factors and to identify bridge factors or common factors between them.

In short, this large-scale PSG study aimed to (1) determine the prevalence of and risk factors for SB and (2) investigate the relationships between RMMAs, arousals, and respiratory events in adults with OSA.

METHODS

This was a prospective cross-sectional study. The protocol was approved by the institutional Medical Ethics Committee of the OLVG West, Amsterdam (WO 16-577). This study has also been registered on https://trialsearch.who.int (NL8516).

Participants

PSG recordings and profiles (see below) of all patients who were referred to the Department of Clinical Neurophysiology, OLVG West, Amsterdam, the Netherlands, between April 2017 and July 2018 were reviewed. Patients who met the following criteria were included in this study: (1) age ≥ 18 years, (2) diagnosed with OSA according to patients' profiles, and (3) AHI ≥ 5 events/h of sleep. Exclusion criteria were (1) total sleep time ≤ 4 hours[28]; (2) continuous artifacts or missing data on electroencephalography, electromyography (EMG), or respiratory channels (eg, airflow, oxygen saturation) of PSG recordings; and/or (3) patients with OSA treatment in situ during PSG.

Patients' profiles

Patients' profiles, including their age, sex, primary diagnosis, secondary diagnoses, comorbidities, medication, and previous treatment history, were collected by one of the authors (A.H.) and colleagues at the Department of Clinical Neurophysiology.

Polysomnographic recordings

A portable PSG system (SOMNOscreen Plus; SOMNOmedics GmbH, Randersacker, Germany) was used to perform a full-night sleep recording. The following channels were recorded: electroencephalography (F4:C4, C4:O2, F3:C3, C3:O1), electrooculogram (E2:M1, E1:M2), electrocardiogram, bilateral masseter muscle EMG, anterior tibialis EMG, pressure airflow, snoring, abdominal and thoracic respiratory effort, oxygen saturation, heart rhythm, plethysmography, and sleep position.

Polysomnographic scoring

Prior to scoring, all PSG recordings were anonymized by removing patients' general information (name, sex, date of birth, and identity number). Subsequently, they were renamed by a series of numbers by a sleep technologist at the Department of Clinical Neurophysiology. Afterward, the PSG scoring was performed offline using DOMINO software (SOMNOmedics GmbH, Randersacker, Germany). Sleep stages and respiratory

events (eg, apnea, hypopnea) were scored manually by certified PSG technicians according to the American Academy of Sleep Medicine (AASM) scoring criteria.[29] Sleep arousals were analyzed according to the AASM scoring manual and were further classified as respiratory arousal and nonrespiratory arousal by 2 of the authors (D.L. and B.K.).[29,30] Arousals occurring at the termination of respiratory events (ie, apnea, hypopnea) were defined as respiratory arousal, while arousals without preceding respiratory events were defined as nonrespiratory arousal.

The EMG signals were filtered between 10 and 100 Hz.[29] A notch filter of 50 Hz was used to remove interference from nearby electrical sources. Also, the electrocardiogram elimination technique was applied to remove electrocardiogram contamination from EMG signals. RMMAs were scored by 2 of the authors (D.L. and B.K.) according to previously reported criteria.[31,32] Each EMG burst had a mean amplitude at least 2 times higher than the baseline EMG amplitude on bilateral masseter EMG traces. EMG bursts occurring within an interval shorter than 3 seconds were defined as a single EMG episode. RMMAs were classified into 3 subtypes: phasic RMMA, tonic RMMA, and mixed RMMA (phasic RMMA: ≥ 3 continuous EMG bursts lasting 0.25–2 s; tonic RMMA: each EMG burst was > 2 s; and mixed RMMA: both phasic and tonic EMG patterns were observed within a single EMG episode). In addition, RMMAs were considered to be related to arousals (respiratory arousal or nonrespiratory arousal) when they occurred within 5 seconds of arousals.[26]

Statistical analysis

Before the start of masticatory muscle activity scoring, 30 PSG recordings were randomly selected to assess interscorer reliability. The interscorer reliability was tested by an average measure, absolute agreement, 2-way mixed-effects model.

The diagnosis of SB was based on an RMMA index of at least 2 episodes per hour of sleep.[31] When the RMMA index was at least 4 episodes/h, the individuals were diagnosed with severe SB. The normality of quantitative variables was tested by the Shapiro-Wilk test. For normally distributed variables, data are presented as means with standard deviations. For non–normally distributed variables, data are presented in quartiles (25%|50% (median)|75%). According to the presence or absence of SB, the entire sample was divided into an SB group and a non-SB group. The comparison of variables between the SB group and the non-SB group was analyzed by independent-samples *t* test, Mann-Whitney *U* test, or chi-square test.

For the first aim, the prevalence of SB was expressed as the percentage of positive SB of the total sample. A binary logistic regression analysis, with SB (positive or negative) as the binary dependent variable, and with age, sex, BMI, sleep- and respiratory-related polysomnographic variables (ie, N1, N2, supine position, AHI, respiratory arousal, nonrespiratory arousal) as the independent variables, was performed to identify the risk factors for SB in individuals with OSA. Although several sleep-related disorders have been reported to be possibly related to SB, only a few of them (eg, insomnia and periodic leg movements during sleep) have been confirmed objectively by PSG studies.[14] However, the case numbers of insomnia and periodical leg movements during sleep in this study were quite small (4 and 2, respectively). Therefore, other sleep-related disorders were not included in the regression analysis for identifying risk factors for SB.

For the second aim, the relationship between RMMAs, sleep arousals, respiratory events (eg, AHI), and other factors was analyzed by a network analysis. The network analysis was performed using the Mixed Graphical Model of the *R* package "bootnet" (Version 1.5) with conditional dependence relationships and network regularization (least absolute shrinkage and selection operator). The estimated relationships represent the unique association between 2 variables after controlling for other variables. The *R* package "qgraph" (version 1.9) was used to visualize the network; all variables were presented as nodes, while the correlations between variables were displayed as edges. Finally, the robustness of the estimated network was analyzed by the bootstrapping method to investigate the network's accuracy. Bootstrapping would repeatedly estimate a model from simulated data (bootstrap = 1,000 samples) and show 95% of bootstrapped confidence intervals. If the 95% confidence interval of an edge does not cover zero, this edge is strong enough to present in the network. The details of the methodology concerning the network analysis have been reported in a previous publication of our research group.[33] The network analysis was performed in R (version 4.1.2; R Core Team, Foundation for Statistical Computing, Vienna, Austria).

In addition, the Wilcoxon signed-rank test was used to analyze the difference between the percentage of RMMA related to sleep arousal and that of RMMA unrelated to sleep arousal, and between the percentages of RMMA related to respiratory arousal and that of RMMA related to nonrespiratory arousal. Statistical analyses, except for the network analysis, were performed using SPSS Statistics (version 26; IBM Corporation, Armonk, NY, USA); statistical significance was determined at $P < .05$.

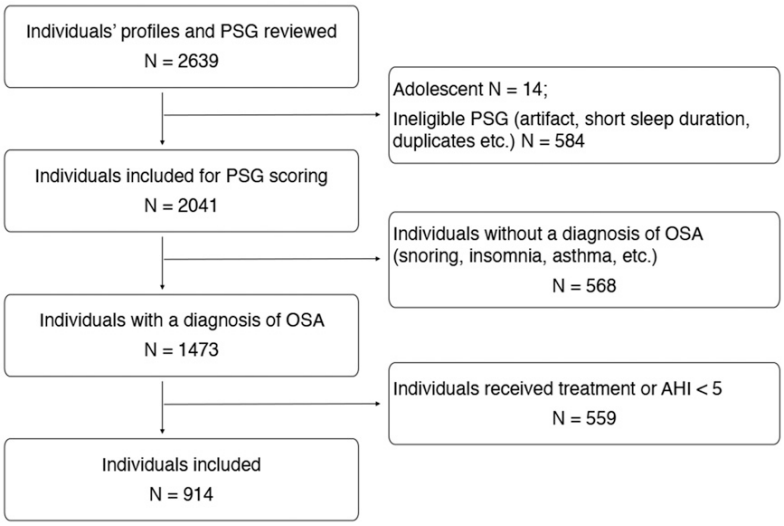
RESULTS

Participants

We reviewed 2,639 patients who were referred to the sleep laboratory. Based on the inclusion and exclusion criteria, 1,725 of them were excluded for various reasons. The screening of patients is shown in **Figure 1**. Finally, 914 patients (305 females and 609 males) were included in this study. Among them, 432 (47.3%) were diagnosed with mild OSA, 252 (27.6%) with moderate OSA, and 230 (25.2%) with severe OSA. The median age of the included participants was 53 years, with an interquartile range of 17 years. The median BMI was 29.1 kg/m^2, with an interquartile range of 6.9 kg/m^2.

Table 1 shows the descriptive information of the total sample, the SB group, and the non-SB group. The SB group had a significantly lower BMI than the non-SB group ($P < .001$). In addition, SB was more prevalent in males than in females ($P < .001$). In addition, there was no significant difference in age between the 2 groups ($P > .05$). Similarly, the AHI and oxygen desaturation index did not show any significant between-group differences. In terms of sleep arousal, the SB group had a significantly higher respiratory arousal index than the non-SB group ($P = .017$), while no such difference was found for the

Figure 1—Flow diagram of participant screening.



AHI = apnea-hypopnea index, OSA = obstructive sleep apnea, PSG = polysomnography.

Table 1—Descriptive variables in individuals with obstructive sleep apnea without and with sleep bruxism.

	Total (n = 914)	Non-SB Group (n = 460)	SB Group (n = 454)	Test Statistics[a]	*P*
Age, y	44.0|53.0|61.0	44.0|53.0|61.0	43.0|53.0|61.0	Z = −1.177	.239
Sex, n					
Female	305	179	126	χ^2 = 12.798	.000*
Male	609	281	328		
BMI, kg/m^2	25.8|29.1|32.7	26.5|29.7|33.6	25.4|28.4|31.6	Z = −4.019	.000*
Total sleep time, h	6.2|7.0|7.8	6.2|7.0|7.8	6.3|7.1|7.8	Z = −0.555	.579
Sleep efficiency, %	82.2|90.1|94.8	82.0|90.1|94.9	82.3|90.0|94.5	Z = −0.267	.790
N1, %	2.6|4.4|7.2	2.7|4.1|6.8	2.5|4.8|7.8	Z = −1.559	.119
N2, %	45.2 ± 10.6	45.2 ± 10.9	45.1 ± 10.4	T = 0.202	.840
N3, %	12.8|18.1|23.7	12.5|18.3|24.1	13.2|17.9|17.9	Z = −0.588	.557
REM, %	17.8 ± 6.5	17.7 ± 6.4	17.9 ± 6.5	T = −0.339	.735
Supine, h	1.0|2.3|3.8	1.0|2.4|3.8	1.0|2.3|3.6	Z = −0.198	.843
Nonsupine, h	3.1|4.5|5.7	3.0|4.5|5.7	3.1|4.6|5.6	Z = −0.408	.683
Total arousal, n/h	5.8|10.3|19.0	4.9|10.2|18.6	6.5|10.4|10.4	Z = −1.681	.093
nRAr, n/h	3.0|5.6|9.7	2.8|5.7|9.7	3.3|5.6|5.6	Z = −0.484	.628
RAr, n/h	1.8|3.9|8.6	1.5|3.5|7.9	2.1|4.1|4.1	Z = −2.396	.017*
AHI, n/h	9.0|15.9|30.4	9.0|15.1|29.8	9.3|16.7|16.7	Z = −0.451	.652
ODI, n/h	12.8|20.7|34.9	12.5|20.4|36.6	13.1|21.0|21.0	Z = −0.128	.898
RMMA, n/h	0.8|2.0|4.0	0.3|0.8|1.3	2.9|4.0|4.0	Z = −26.171	.000*

Data are presented as mean ± SD for normally distributed variables and 25%|median|75% for non–normally distributed variables. [a]Independent-samples *t* test for normally distributed data, Mann-Whitney *U* test for non–normally distributed data, chi-square test for categorical data. *Statistically significant at $P < .05$. AHI = apnea-hypopnea index, BMI = body mass index, N1–N3 = non–rapid eye movement stage 1–3 sleep, nRAr = nonrespiratory arousal, ODI = oxygen desaturation index, RAr = respiratory arousal, REM = rapid eye movement, RMMA = rhythmic masticatory muscle activity, SB = sleep bruxism.

nonrespiratory arousal index (P = .628) and for the total arousal index (P = .093). Additionally, total sleep time, sleep efficiency, sleep position duration, and percentages of sleep stages were similar between the 2 groups (all P > .05).

Prevalence of and risk factors for SB

An excellent interrater agreement was achieved for RMMA scoring (0.925). Of the 914 adults with OSA, 454 (49.7%) were diagnosed with SB and 223 (24.4%) were diagnosed with severe SB.

Table 2 shows the outcomes of the binary logistic regression. Compared with females, males had a significantly higher risk of having SB (odds ratio [OR] = 1.425, P = .005). Lower BMI (OR = 0.951, P = .000) and a higher percentage of N1 sleep (OR = 1.032, P = .042) significantly increased the odds of having SB. There were no significant associations between SB and age, AHI, respiratory arousal, nonrespiratory arousal, and the duration of supine position in the OSA population.

Associations between RMMAs, arousals, and respiratory events

Figure 2 shows the visualization of the network analysis. As presented in the figure, the RMMA index has a negative correlation with BMI and a positive correlation with male sex. No direct association was found between RMMA and respiratory events. However, both RMMA and AHI were correlated with BMI, suggesting an indirect correlation between RMMA and respiratory events. In addition, neither respiratory arousal nor nonrespiratory arousal had a direct association with RMMA. In addition, the correlation between SB and N1 sleep that was shown in the logistic regression analysis did not present in the network analysis after controlling for all the other factors. The bootstrapped confidence intervals of the network model are presented in the supplemental material (**Figure S1** in the supplemental material).

In addition, the majority of RMMAs (median = 85.7%) were time-related to sleep arousals, which was significantly higher than the percentage of RMMAs unrelated to arousals (median = 14.3%, P < .001). Further, more RMMAs were related to nonrespiratory arousal than to respiratory arousal (46.8% vs 25.0%; P < .001).

DISCUSSION

This large-scale PSG study aimed to determine the prevalence of and risk factors for SB in adults with OSA and to investigate the associations between RMMAs, arousals, and respiratory events in adults with OSA. Based on our results, 49.7% of adults with OSA had comorbid SB. Male sex, lower BMI, and a higher percentage of N1 sleep significantly increased the risk of having SB. Further, RMMA did not have a direct association with respiratory events and sleep arousals; however, most RMMAs were time-related to arousals.

Prevalence of and risk factors for SB in adults with OSA

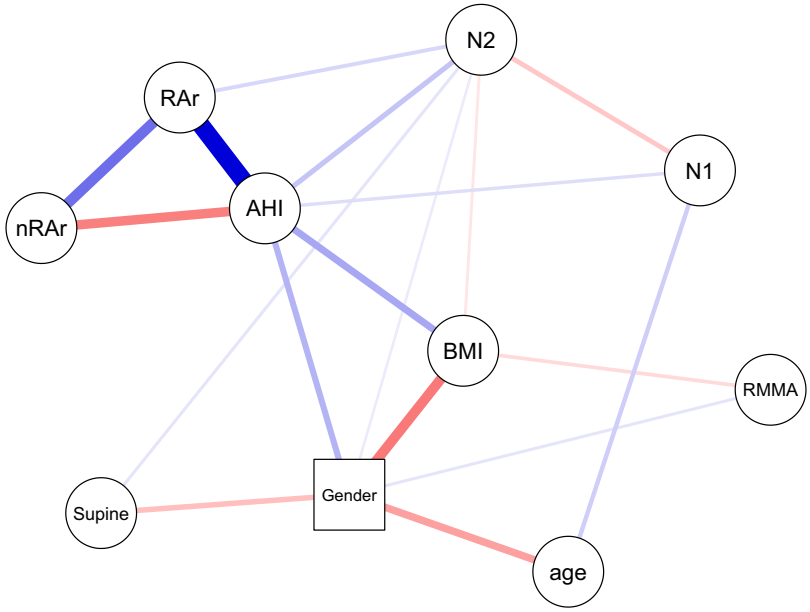
The present PSG study with a large-scale sample confirmed that nearly half (49.7%) of adults with OSA had comorbid SB. This result confirmed that SB is a common comorbidity of OSA that demands close attention. Further, it is important to note that the prevalence is much higher than that from studies in which SB was measured by self-report. As reported by a previous study, the prevalence of self-reported SB in individuals with OSA (n = 300) was 26%.[15] Another study showed that the prevalence of SB in OSA was 27.5% based on self-report, while in the same study, the prevalence became 52.4% when using PSG to measure SB.[34] These studies suggest that self-report or questionnaires may underestimate the prevalence of SB in the OSA population. Further, the underestimation of SB due to the use of self-report suggests that a large number of individuals with OSA are unaware of SB. With this, more attention on the negative consequences of SB is demanded from sleep doctors, dentists, as well as from the individuals with OSA themselves.

Table 2—Binary logistic regression model of factors related to sleep bruxism in adults with OSA.

Predictors	β (SE)	OR	95% CI for OR	*P*
Age	−0.009 (0.006)	0.991	0.980–1.002	.113
Sex				
Female	Reference	–	–	
Male	0.354 (0.153)	1.425	1.055–1.924	.021*
BMI	−0.050 (0.013)	0.951	0.926–0.976	.000*
N1%	0.032 (0.016)	1.032	1.001–1.064	.042*
N2%	−0.005 (0.007)	0.995	0.982–1.009	.510
Supine	0.014 (0.036)	1.015	0.946–1.088	.687
Respiratory arousal	0.001 (0.010)	1.001	0.981–1.021	.954
Nonrespiratory arousal	−0.002 (0.014)	0.998	0.971–1.025	.868
AHI	0.003 (0.005)	1.003	0.992–1.014	.586

*Statistically significant at P < .05. AHI = apnea-hypopnea index, BMI = body mass index, CI = confidence interval, OR = odds ratio, OSA = obstructive sleep apnea, SE = standard error, β = regression coefficient.

Figure 2—Network model of sleep bruxism in adults with obstructive sleep apnea.



The squares represent categorical variables; the circles represent continuous variables. The blue lines represent positive associations; the red lines represent negative associations. Thicker and darker colored lines refer to stronger associations. AHI = apnea-hypopnea index, BMI = body mass index, nRAr = nonrespiratory arousal. RAr = respiratory arousal. RMMA = rhythmic masticatory muscle activity (biomarker of sleep bruxism).

Most studies that investigated the risk factors for SB did not show a significant difference between males and females in the prevalence of SB in the general population.[22,35] However, based on our analysis, male sex was a significant risk factor for SB in the OSA population (OR = 1.503), which is in line with another study that was composed of adults with OSA.[18] These contrary findings may due to the population that was under investigation. Male sex has been proved to be an independent risk factor for OSA.[36] In addition, previous studies considered SB to be secondary to sleep arousal.[27,37,38] At the same time, in individuals with OSA, males not only have more sleep arousals than females but also have a higher ventilatory response to sleep arousals, which might be related to the inherent sex differences in, among others, the collapsibility of the upper airway, the neurochemical control mechanisms, and sex hormones.[39,40] Thus, the higher frequency of and response to sleep arousals in males than in females could support that male sex is a risk factor for SB in adults with OSA.

Our results showed that patients with a lower BMI had a higher risk of experiencing SB (OR = 0.954, $P < .001$). This is in accordance with the results of another large-scale PSG study (n = 1042).[41] That study reported that, in the general population, individuals with normal weight had a significantly higher frequency of SB than those with obesity.[41] After controlling for other factors by the network analysis, RMMA also showed a negative correlation with BMI. However, the underlying mechanism for this association is still unclear. It may be attributed to the deleterious effects of obesity on skeletal muscle structure and performance, such as physical inactivity and inflammatory changes.[42,43] Future studies are warranted to investigate the underlying mechanism.

Previous findings suggest that most RMMAs and arousals occur in sleep stages 1 and 2, but rarely in stage 3 and rapid eye movement (REM) sleep.[44] The regression analysis in this study revealed that a higher percentage of sleep stage 1 increased the odds of SB. However, the percentage of N1 and N2 sleep in this study was within the normal range[45] and the OR for the percentage of N1 sleep is close to 1.0 (OR = 1.032). Moreover, the results of the network analysis showed that there is no (in-)direct association between SB and N1 and/or N2 sleep. This is consistent with previous studies that showed that individuals with SB display normal sleep architecture in terms of total sleep time, sleep latency, and percentage of the sleep stage distribution.[46,47] All of this evidence together suggests that the percentage of N1 sleep may not be clinically relevant to SB.

In summary, SB is highly prevalent in adults with OSA. It is recommended for sleep doctors to carry out a routine screening and monitoring of SB for adults with OSA, particularly for those with male sex and a lower BMI. It would be better to refer those who report complaints about SB or show apparent negative consequences of SB (eg, severe tooth wear and/or orofacial pain) to dentists for further examination and collaborative management. For individuals with OSA and SB, although oral appliances could relieve both OSA and SB in some cases, the elevated likelihood of oral appliance breakage should also be considered.

Association between RMMA, arousal, and respiratory events

As presented in **Figure 2**, RMMA did not directly correlate to AHI. Moreover, some studies on the temporal relationship

between RMMA and respiratory events demonstrated that a large amount of RMMAs were time-unrelated to respiratory events.[48,49] These results are in line with some other studies that showed that masticatory muscle activities are more likely related to respiratory arousals rather than the respiratory events per se in individuals with OSA.[25,26] Moreover, our results showed that more RMMAs were related to nonrespiratory arousals than to respiratory arousals. All of these findings suggest that the occurrence of RMMA seems not to rely on the presence of respiratory events. However, these findings cannot preclude the possibility that RMMA is related to a specific type of respiratory event. Thus, future studies are needed to investigate the associations between RMMAs and different types of respiratory events in individuals with OSA and central sleep apnea.

Although RMMA is suggested to be related to sleep arousals, no direct link between RMMA and sleep arousals (including respiratory arousal and nonrespiratory arousal) was found from the network analysis. These results suggest that there is not a linear correlation between the RMMA index and sleep arousal index. However, it is important to note that neither the regression analysis nor the network analysis takes the temporal relationship between variables into consideration.[14] Based on our results, most RMMAs (85.7%) were time-related to sleep arousals. Further, the proportion of RMMA in relation to sleep arousals in our sample—individuals with OSA—is quite close to that in individuals with SB without OSA (88%).[38] These results suggest that SB/RMMA is an arousal-related autonomic motor response during sleep without differences between individuals with or without OSA. Therefore, these seemingly contrasting findings regarding the relationship between SB/RMMA and arousal support the theory that arousal only acts as a permissive window for the occurrence of RMMA rather than as a generator.[50]

Furthermore, with regard to the association between OSA and SB, it could be that OSA characterized by frequent sleep arousals provides more chances for the occurrence of SB. It is of importance to note that our findings cannot preclude other possible mechanisms for the association between OSA and SB. For example, studies have reported that some neurochemicals with direct activity in respiratory muscles' motor nuclei and arousal systems, such as glutamate, glycine, serotonin, acetylcholine, and gamma-aminobutyric acid, have also been reported to be related to the genesis of SB.[51,52] Therefore, OSA-related factors that influence the secretion or metabolism of these neurochemicals may also play an important role in the occurrence of SB in OSA.

Strengths and limitations

This study has several strengths. First, this study was performed in a large sample of individuals with OSA, which ensures the statistical power and reliability of our results. Second, compared with univariate analysis and logistic regression analysis, network analysis takes all variables into account in a single model, which controlled for the influence of other covariates on the association between pairwise variables. Also, the network analysis and its graphical representation showed direct and indirect associations between variables, which helps in understanding the intertwined correlations between factors, and to identify the independent risk factors for RMMA and SB.

Apart from the strengths, several limitations should be kept in mind during the interpretation of the results. First, although SB was assessed objectively by PSG, the absence of audio and video recordings may, to some extent, overestimate the RMMA index.[31] Nonetheless, as reported by previous studies, the accuracy of RMMA scoring with PSG systems without audio and video remains relatively good for research and clinical aims.[31,53] Moreover, the prevalence of SB in adult patients with OSA found in this study is similar to that of previous PSG studies that had audio and video,[4,18] suggesting that our results remain reliable. Second, we did not perform self-report and clinical inspection in this study. It is of importance to acknowledge that, although PSG is currently considered the gold standard for SB assessment, the RMMA index itself can hardly link to the clinical outcomes of SB without the information of self-report and clinical inspection.[54,55] Future studies based on a standardized scoring system using SB metrics that are relevant to the negative consequences of SB will help identify risk factors for SB of high clinical relevance. Third, the participants of this study were those who received PSG recordings in a hospital setting. These individuals were likely to have substantial signs or symptoms of OSA for which they sought treatment. Thus, the OSA sample might deviate from a representative OSA group in the general population. This may have influenced the outcomes of this study. Future research is suggested to include a general population–based OSA sample.

CONCLUSIONS

This study demonstrated that nearly half of patients with OSA have comorbid SB. Male sex, lower BMI, and a higher percentage of sleep stage 1 increase the odds of having SB. However, the clinical relevance of the latter is doubtful given the low OR and lack of other supportive evidence. Further, although SB was not directly correlated with respiratory events and sleep arousals, the majority of SB events were time-related to sleep arousals.

ABBREVIATIONS

AHI, apnea-hypopnea index
BMI, body mass index
EMG, electromyography
OR, odds ratio
OSA, obstructive sleep apnea
PSG, polysomnography
RMMA, rhythmic masticatory muscle activity
SB, sleep bruxism

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Address correspondence to: Deshui Li, DDS, MSD, Department of Orofacial Pain and Dysfunction, Academic Centre for Dentistry Amsterdam (ACTA), Gustav Mahlerlaan 3004, 1081 LA, Amsterdam, The Netherlands; Email: d.li@acta.nl

DISCLOSURE STATEMENT

All authors have seen and approved the manuscript. Work for this study was performed at OLVG West and Academic Centre for Dentistry Amsterdam (ACTA), Amsterdam, The Netherlands. The authors report no conflicts of interest.